

The University of Chicago

PRODUCTION OF REFLEX PRESSOR
AND DEPRESSOR RESPONSES BY SELECTIVE
ELECTRICAL STIMULATION
OF THE VAGUS

A PART OF A DISSERTATION SUBMITTED TO
THE FACULTY OF THE DIVISION OF THE
BIOLOGICAL SCIENCES IN CANDIDACY FOR
THE DEGREE OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF PHYSIOLOGY

1934

By

WADE HAMPTON MARSHALL

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The reflex vasomotor and, in an incidental manner, the respiratory reflexes associated with central stimulation of the sectioned vagus have been investigated with the purpose of determining to what extent the acute mammalian preparation may be standardized as regards the behavior of vasomotor and respiratory centers. To this end certain general conditioning factors such as type and degree of narcosis, carbon dioxide tension, venous return, etc. must be considered first; then varying certain of these factors and following the reaction by changes of reflex response to electrical stimulation of afferent nerves, particularly if the electrical responses in the central nervous system are followed simultaneously, constitutes a reasonable approach to these problems.

Electrical stimulation of the central end of the sectioned vagus seems to have yielded uncertain results in the dog, cat, and rabbit as regards consistent production of definite pressor and depressor effects. Experimenters have observed more or less retained pressor reactions over apparently wide ranges of stimulation in these animals, and under special conditions even in the case of the depressor nerve of the rabbit. Stewart and Pike (1907) found a rise in pressure on central stimulation of the depressor in the rabbit and the vagus in the cat as the preparations recovered from prolonged cerebral anemia. Hooker (1907) found that, when the heart was first slowed by peripheral vagal stimulation, central stimulation produced a rise in blood pressure with and without cardiac acceleration. In some instances this conditioning factor was not needed to demonstrate the pressor response. Bayliss (1908) described an apparent reversal of response in the depressor action after injection of strychnine. Langley (1912, 1919) considered this reversal to be due to an alteration of vasomotor centers, which led to a decrease of response to depressor nerves and an augmented response to the pressors. Scott (1924) secured a reversal by directly injuring the depressor points (Ranson and Billingsley, 1916) on the floor of the fourth ventricle. More recently Bishop, Heinbecker, and O'Leary (1933) have conducted general examinations of the fiber groups and functional relations of the vagus nerve.

Method

The electrical stimulator was a thyatron-vacuum tube device providing the three independent and quickly variable parameters of frequency, intensity, and duration of each impulse. The principle of operation is the amplification of a condenser discharging voltage through a screen grid stage of Class A amplification, the output of which is battery-coupled to two triode tubes operated in parallel as Class C amplifiers. The latter are completely saturated with respect to the grid voltage/plate current curve, as long as the thyatron is discharging the condensers. As the plate current of the triodes is reduced to zero by a highly negative grid bias, the output consists of nearly rectangular pulses (Fig. 1, 12) of a duration of 0.02 to 3.0 or more sigma, depending upon capacity of condensers C_1 , C_2 , C_3 , or C_4 .

The wiring diagram is shown in Figure 1. In order to secure simplicity of presentation some panel controls have been omitted in the diagram.

When plug "p" is removed from JR8 the unit delivers condenser discharges, as shown in Figure 1, 12. When plug "p" is inserted in JR8 the rectangular impulses are produced as shown in Figures 3 and 6. Duration is controlled by condenser settings, C_1 , C_2 , C_3 , C_4 , intensity by potentiometer outputs, numbers 1, 2, and 3, and frequency by an entirely separate thyatron discharge unit (not shown in the diagram), which drives the stimulator through an air core grid coupling transformer ("Tr." on the figure). The grid voltage on the thyatron and the plate resistance are rather critical and must be checked, with output connected to an oscillograph, every few hours. In practice the output is switched to the oscillograph frequently during the experiment. If desirable, the oscillograph--if it is an electrostatic device such as a cathode ray--may be connected across the output potentiometer during the course of the experiments. A circuit plug is inserted in J15, as this unit of the circuit is not used.

The stimulating potential was applied through silver electrodes plated with silver chloride.

Stimulation with "galvanic" stimuli has certain distinct advantages. Bishop (1929) describes the generally accepted conditions of nerve excitation as "stimulation of a nerve takes place in accordance with laws governing charging of a condenser or polarizing of a membrane." Bishop (1928a) has further shown that this process is clearly demonstrable by superimposing an induction shock on galvanic currents at varying strengths and durations of the latter. The strength of the induction shock required

to stimulate the polarized nerve decreases in an exponential fashion with increased duration of the polarizing or galvanic potential. It is obvious that, particularly for smaller nerves of higher thresholds, a sustained potential would probably be more satisfactory than a rapidly falling potential from a condenser or induction coil discharge. It would appear that a more ideal method would be to apply a voltage that increased along an exponential curve with time paralleling the increasing polarization potential of the nerve, such as used by Bishop and Erlanger.

It is of further distinct advantage to be able quickly to vary frequency, intensity, and duration, as a means of discriminating fibers on a functional basis. The low threshold fibers can be separated from the high threshold fibers on a basis of the parameters, by appropriate adjustment of all three, since low threshold fibers will respond at a high frequency, and, in general, evoke maximal reflex response only at a high frequency of stimulation; while high threshold fibers will give a maximal effect with a relatively much lower frequency. Thus disregarding the factor of repetitive discharge (Bishop, 1928b), it is evident that the high potential low frequency stimulation will favor response of high threshold fibers. The factor of galvanic suppression of low threshold fibers with high potential galvanic stimulation may also be significant in suppressing the low threshold response.

However, the polarization factor must be recognized as undesirable, particularly as the output circuit of the stimulator must be of several hundred ohms resistance, through which the nerve can not depolarize as readily as through the low ohmic resistance of an induction coil. Presumably this may be of some significance. Actually its effect was not apparent in the experiments cited below.

Procedure

Dogs were anesthetized with nembutal, barbital, and combinations of morphine and nembutal, with ether applied when necessary in any case. The cats were anesthetized with nembutal. Blood pressure was taken from the carotid artery, or more often from the femoral artery. Simultaneously respiration records were taken from the chest and occasionally from the abdomen also. One experiment was performed on a dog with the chest open.

The central vagi were stimulated at frequencies varying from 1 to 200 per second, at intensities of 0 to 30 volts, and durations of 0.02 to 10 sigma. The electrodes were usually about 1 cm. apart. It must be recognized that the electrical quantities,

particularly intensity, are only of relative significance when nerve trunks of different physical dimensions are compared. Flattening of the electrodes with silver chloride was found to be rather important in suppressing anodal block.

Results

A high threshold pressor response was regularly found in dogs (10 experiments). A good example of the intensity relation is shown in Figure 6a, with stimuli of 3 sigma duration and frequency of 20 per second. The relative intensities are approximately 1, 1.5, 3.6, and 16.6. The secondary phase in the blood pressure fall may be due to resumption of respiration or to secretion of adrenalin (Houssay and Molinelli, 1927).

The effect of maintaining a constant intensity and frequency and varying the duration is shown in Figure 6b. The durations used were 0.4, 1.6, and 3.0 sigma, and the intensity in units equal to those in Figure 6a was 13.0, while frequency was the same, 20 per second.

The depressor effect can always be separated from the pressor effect in the dog. It is elicited best with a frequency of 50 to 100, and duration of 0.15 to 0.2 sigma and a relative intensity of about 0.3. A discrimination of these effects by differences in duration of each impulse in the same dog is shown in Figure 6c. Frequency is 5 per second, intensity 1.3, and duration 0.2 sigma for the depressor and 3 sigma for the pressor action. In this specimen the respiratory behavior was anomalous, for usually any stimulation of the dog vagus sufficient to produce a depressor effect will inhibit respiration, and also, as Erlanger (1926-27) observed for other afferent nerves, strong stimulation causing increased blood pressure often causes acceleration of respiration. Figure 6c shows depressor-pressor discrimination with another preparation by increase of intensity, frequency, and duration remaining constant.

In nine out of ten experiments on dogs, the left vagus was regularly more potent than the right, and the heart rate was usually accelerated up to 20 per cent by strong central vagal stimulation (with both vagi cut).

In one experiment on the effect of salt concentration within the cisterna magna, it was found that the strongest stimulation produced only a slight elevation of blood pressure. Weaker stimulation caused a fall in pressure and extreme slowing of the heart, for instance decreasing the rate from 2.3 to 1.1 per second in one case. Occasionally the heart was completely stopped for two or three beats. After cutting the right vagus, the heart rate

increased more than is usually the case upon removal of vagal stimulation. Weak stimulation of the central end of the right vagus now produced a very abrupt rise in blood pressure of over 100 per cent. Presumably this dog's left vagus contained a great majority of pressor fibers.

A record from another experiment, in which both vagi were cut, is shown in Figure 7a, in which the intensity was high, frequency 20 per second, and the depressor and pressor effects separated by varying duration from 0.02 to 0.12 sigma. The depressor action also slowed the heart from 2 beats per second to 1.6 per second. The pressor action accelerated the heart from 1.8 beats to 2.2 per second. The pressor effect is always well maintained during stimulation of the nerve (up to two minutes, at least), while the depressor effect is never maintained as shown here (Fig. 7a). Another example from the same dog is shown in Figure 7b, illustrating the greater potency of the right vagus. The heart block here is only occasionally seen.

Long descending phases of the blood pressure curve after strong stimulation were sometimes observed (Fig. 8a), which disappeared after the adrenals were ligated (Fig. 8b).

Another example of the depressor and pressor effects are shown in Figures 9a and 9b. The respiration effect shown here is more typical for stimulation which is adequate to produce a strong pressor response than is shown in the preceding tracings. Usually, however, the depressor action is accompanied by inhibition of respiration. The pressor effect is sometimes followed by a short fall below normal. Usually this is found with weaker stimulation, stronger apparently calling forth adrenalin secretion. In one case (Fig. 9c) the pressure was elevated about a centimeter for 65 seconds by a frequency of 10 per second, a duration of 0.02 sigma, and a high intensity. The fall occurring after cessation of stimulation was quite pronounced and the normal level was reached after 30 seconds. With the duration increased to 1.6 sigma, however, the blood pressure slowly fell to normal, instead of quickly falling below normal. This is a striking parallel to the case of the cat to be discussed presently. This effect is not always seen.

A further typical example of the pressor response from another dog is shown in Figure 10a.

One experiment was performed to see if some physiological function might be demonstrated, as well as to ascertain whether all the pressors come from the region of the heart. Figures 11a (1) and 11b (1) show the result of stimulating the left vagus below the heart. The slow rate of rise, illustrated in Figure 11a (1), or the initial fall, as in Figure 11b (1), accompanying

stimulation of the vagus below the heart, is in each case undoubtedly due to obstruction of venous return during the adjustment of the electrodes, but the pressor effect was quite definite. Stretching the walls of the opened abdomen produced a pressor effect diagrammatically quite similar to the electrical stimulation of the vagus, shown in Figure 11a (2). Moderate distention of the stomach with air produced a similar effect, shown in Figure 11c (1) and (2), though the effect on venous return was not controlled and constitutes an objection to this particular observation. Gentle massage of the stomach walls, in such a way that the venous return could not possibly be affected, produced a similar result, shown in Figure 11c (3) and (4). While only one experiment of this nature was performed, the results were sufficiently definite to warrant the conclusion that some of the end organs of the pressor fibers are in the stomach walls, and that the rise in blood pressure and acceleration of the heart accompanying strong hunger contraction (Carlson, 1913) may be mediated by the left vagus. It is also probable that some of the respiratory effects on blood pressure are mediated by similar end organs in the diaphragm and abdominal walls.

Several experiments on the effect of the salt concentration within the cisterna magna have yielded interesting results. We have adopted the technique described by Jacobs (1933) for continuous intravenous injection to fluid replacement within the cisterna magna. A small rubber tube is injected into the cavity through a Number 16 hypodermic needle, which is then removed. The tube will slowly be drawn into the muscle seams, but for many hours it may serve as a communication through which the cerebrospinal fluid may be removed and replaced, even in a normal un-anesthetized animal.

If the calcium ion concentration was lowered by injecting a "balanced" solution containing no calcium, the animal underwent a characteristic muscular spasm, accompanied by dyspnoea and, usually, by a slight elevation of blood pressure. The blood pressure elevation was usually no larger than could be attributed to peripheral factors associated with the spasm. This general condition was not produced by pressure changes within the cisterna magna, as was shown by control experiments on all preparations with normal solutions. The usual procedure was to withdraw 3-5 cc. of cerebrospinal fluid and replace it with an equal volume of balanced salt solution. This procedure was always repeated at least three times in the course of each replacement. The spasm appeared very quickly and lasted from 3 to 15 minutes, depending roughly on the degree of the calcium depletion.

The vasomotor reflex effects attending central stimulation with lowered calcium ion concentration were investigated in four preparations. In general, it was found that low calcium ion concentration tended to decrease depressor effects and to increase pressor effects, while respiration inhibition was definitely less complete for low and moderate intensities of stimulation, and was augmented more than normally by strong stimulation.

When a few cubic centimeters of the cerebrospinal fluid were replaced by an equivalent volume of a solution containing sodium citrate, which produced a sudden marked lowering in calcium ion concentration, a more violent reaction was obtained. The results of two such experiments are shown in Figures 4 and 5. In the first experiment, the calcium was lowered by the injection of the citrate solution from a concentration artificially made higher than normal. Within 45 seconds after the citrate injection, a stimulation which formerly gave a depressor effect now produced a definite pressor response (compare Figs. 4 1 and 3). The respiration effects were not distinctly changed within this period. Stimulation which gave a clear pressor action with high (and also with normal) calcium, and which completely inhibited respiration, as shown in Figure 4 (2), produced in this animal after citrate injection a higher peak value, but did not definitely increase the pressor action, illustrated in Figure 4 (4), while respiration was not completely inhibited after the citrate injection.

Another preparation gave the results shown in Figure 5, where (1) and (3) represent pressor reactions to the same values of stimulation before and after citrate injection, and (2) and (4) the depressor response under the same conditions. The increased magnitude of the pressor response is quite definite. The depressor response was apparently reversed also, but it is to be noted that respiration was not stopped after the citrate injection; so the blood pressure curve must be interpreted accordingly. The first preparation, illustrated in Figure 4 (2) and (4), showed this reversal of the depressor response more clearly, with respiration near zero; so the evidence points toward a definite reversal not associated with respiration effects.

The experiments indicate either a reversal of response to stimulation of the same fibers brought about by a change in calcium ion concentration, or, what is more reasonable, they indicate the presence of low threshold pressors which are revealed only when the depressor effect is inhibited. The nature of the inhibition, depressor action, and augmentation of pressor action is probably central, but until the indirect effects from the general muscular spasm and increased muscle tone are eliminated, the decision cannot be made.

Similar to the experience with others, the experiments with cats were not consistent. The presence of low threshold pressors in the vagi could be readily demonstrated, but the magnitude of elevation was unpredictable (Fig. 12a; Figs. 17a and 17b).

In one case a separate nerve was found in the left carotid sheath, which, when separated from the sympathetic and vagus trunks and sectioned, produced a pure pressor effect for stimuli of very low strength (Fig. 14a). In this instance the blood pressure rose from 5.9 cm. of mercury to 7.3, reaching a maximum in 35 seconds, then slowly fell for 20 seconds while stimulation was continued. Seventy seconds after cessation of stimulation the blood pressure had fallen to 5.9 cm. of mercury. The latent period, as estimated to the first definite inflection of the curve, was about 6 seconds, and the heart rate was increased from 2.6 beats per second to 2.8 per second. Respiration was only slightly altered; the first cycle was prolonged about 70 per cent, succeeding cycles only slightly so, and the rate was slightly decreased. The latent period of the respiratory responses was much shorter than that for the vasomotor response. All vasomotor and respiratory responses to stimulation of this separate nerve were similar to this which has been described for a large range of frequencies, durations, and intensities.

The second trunk in this sheath was larger than the first section described above, and about the same size as the vago-sympathetic, which nerve it appeared to be. The vago-sympathetic is always easily discerned and separated in the vagus sheath of the cat. Central stimulation of this nerve produced a slight rise of blood pressure (0.2 to 0.4 cm.). The frequency of respiration was not altered, but the amplitude and total area of each cycle were slightly decreased.

The vagus nerve trunk, the third section in this sheath, gave only a fall of blood pressure on central stimulation. For instance, stimulation for 17 seconds evoked a fall of 2 cm. of mercury in the blood pressure (Fig. 15a). The latent period was about 2.5 seconds, and the fall continued for about 10 seconds after cessation of stimulation, after which the blood pressure slowly returned to normal. The low return to the normal level was usually seen in all cases studied, only one cat showing a quick return (Fig. 16a).

Respiration was usually completely inhibited for moderate values of stimulation.

Six cats were carefully examined for the presence of this third component in a dissectible position in the vagus sheath, but it was not found again. A similar anomaly has been recorded by Scott (1924).

The low threshold pressor effect was not usually found in the vagus trunk, and when found was small (Fig. 12a). The high threshold pressors were usually seen either as pure pressor effects (Figs. 17a and 17b), or more often as pressor followed by depressor effects (Fig. 18a). The higher frequency produced a more immediate response, while the lower frequency sometimes caused either a delay of depressor response or an elevation of pressure during the time of stimulation, illustrated in Figure 19b. It is to be noted that this pressor-depressor effect is independent of mechanical respiration factors, as is shown by comparison of Figures 19a and 19b. In this preparation the pressor-depressor action was quite diagrammatic, the pressor effect having a short latent period and disappearing almost immediately after removal of the stimulus, while the depressor effect persisted as does a long after-discharge. In other preparations, the secondary fall often occurred (Fig. 18a), but the curve for the entire cycle of events was much smoother. A somewhat similar pressor-depressor effect was often seen in dog preparations, as is shown in Figure 21a, in case adrenalin was not called forth by the stimulation, as in Figures 8a and 8b.

In all the cats studied, except the one in which a pure pressor trunk was isolated, central stimulation always produced a more prolonged depressor than pressor effect, as shown in the above-mentioned figures. One extreme example of this may be seen in Figure 19c. The right vagus usually gave a simpler type of response, with slight or no elevation preceding the descent (Fig. 16a). Wright (1928) found somewhat similar results using inductorium stimulation.

It is evident that pressor and depressor effects can usually be adequately separated in "normal" nembutalized cat preparations, using "galvanic" stimuli, but the distinction is not so easily demonstrated as in the dog. Perhaps this difficulty is due to differences in anesthetic behavior. Decerebrate preparations have not been studied in either case.

Summary

These experiments clearly show that functional discrimination of vagus afferents can be made with some degree of certainty, particularly if the frequency of stimulation can be conveniently varied over a wide range. In any case it appears that the fibers are not sufficiently different to study either effect without consideration of the other. The stimulation of the central connections in the medulla is undoubtedly some sort of algebraic resultant of antagonistic control, as advocated by Bayliss (1923), which

may be shifted toward depressor or pressor directions as shown in the experiments cited above. The short latent period and quick disappearance of the pressor response, as compared with the depressor response, and particularly the long after-discharge of the depressor centers following pressor effects in dogs and cats--particularly in the case of the left vagus of the cat--are of vital interest in interpreting the antagonistic control of the central vasomotor system, and suggests reciprocal control in vasomotor centers.

In these experiments good examples of low threshold pressor effects were not seen, and the general frequency relation reported by Gruber (1917) for other afferent nerves, and quite recently reconsidered by O'Leary, Heinbecker, and Bishop (1934) was not thoroughly tested. This may account for the failure to demonstrate the low threshold pressors.

Conclusions

An electrical stimulator has been described with which "galvanic" stimulation can be applied with the three parameters of frequency, intensity, and duration of each impulse quickly and conveniently variable.

Afferent pressor fibers of a threshold range extending from a value near or below that of the depressor fibers to much higher threshold values have been demonstrated in the dog and cat.

Depressor response dominates the lower threshold pressor response, as a rule, in the vagus trunks of the normal anesthetized preparations of the cat and dog. For high intensity, low frequency stimulation, the pressor effects always dominate in the dog; while in the cat--at least for the left vagus--the high threshold pressors tend to dominate for the duration of the stimulus, the depressor response, following as a long after-discharge.

In most dogs the left vagus has a greater pressor effect than the right; while in cats a more nearly pure depressor response is obtained from the right vagus. It would appear that some of the pressor afferents actively inhibit the depressor centers, but that the inhibition has a shorter latent period and a shorter after-discharge than the depressor response.

One anomalous cat was found in which a separate pressor nerve, with a latent period of 6 to 7 seconds, was found in the left vagus sheath.

Decreased calcium concentration in the cisterna magna results in a tendency toward a reversal of depressor response and augmentation of pressor response.

One experiment indicated presence of pressor end organs in the walls of the stomach.

Acknowledgments

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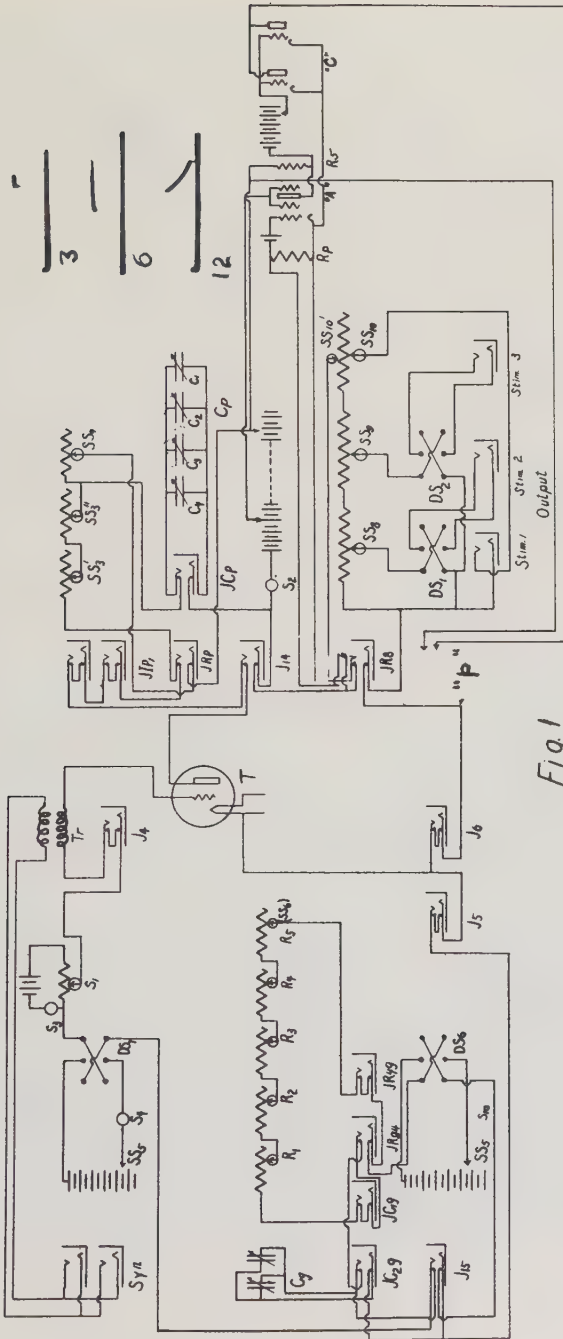
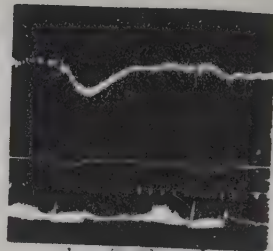
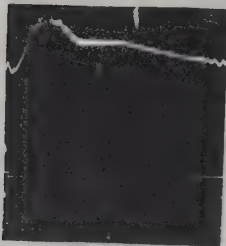
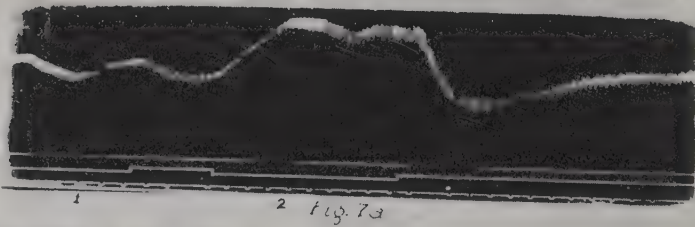
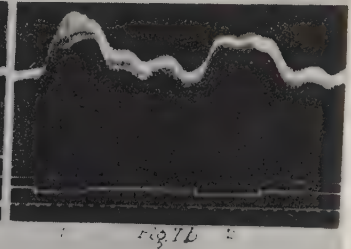
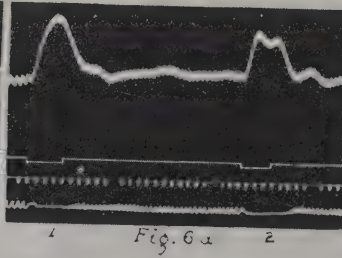
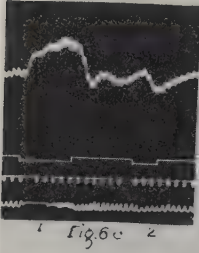
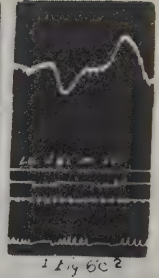
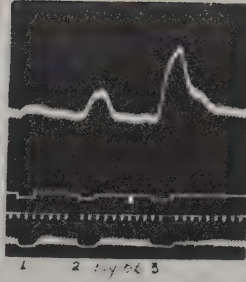
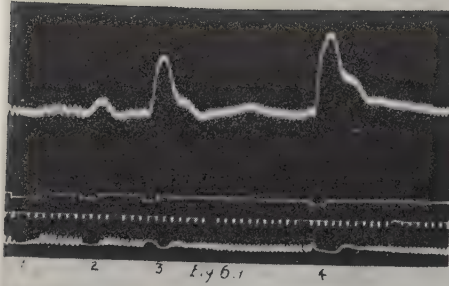


Fig 1



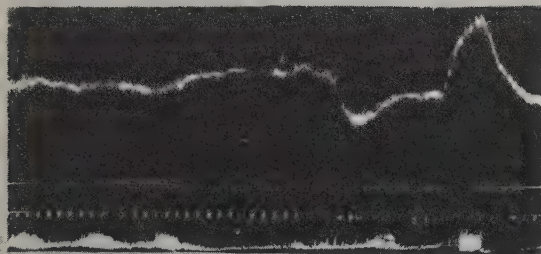


Fig. 9c

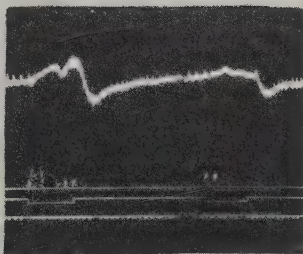


Fig. 9d

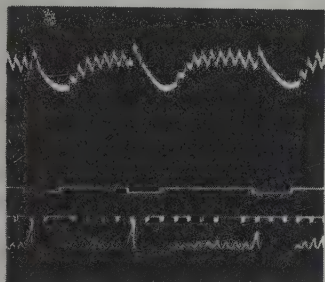


Fig. 9a

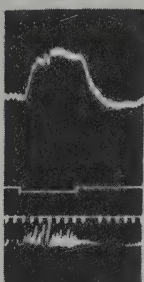


Fig. 9b

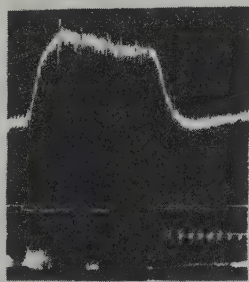


Fig. 9e

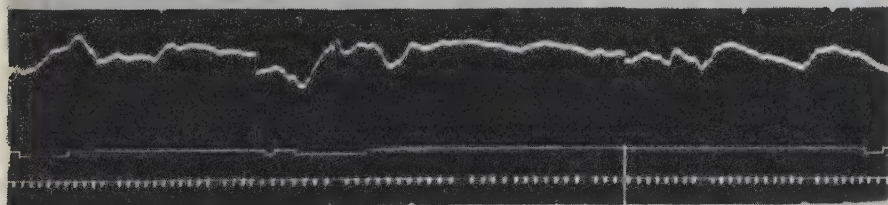


Fig. 11a



Fig. 4

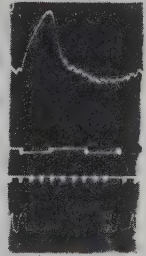
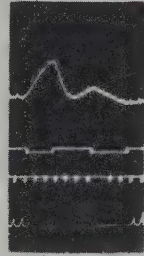


Fig. 5

Fig. 11c



Fig. 18a



Fig. 18c

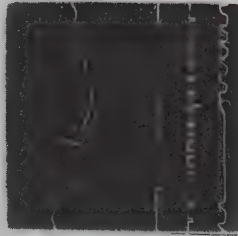


Fig. 19a



Fig. 19c



Fig. 17b



Fig. 17c

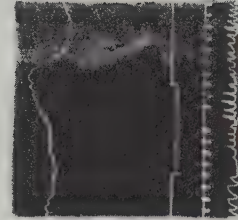


Fig. 19b

MO SEC



Fig. 19d

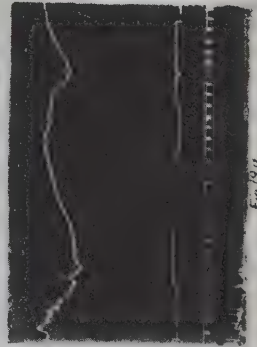


Fig. 19e

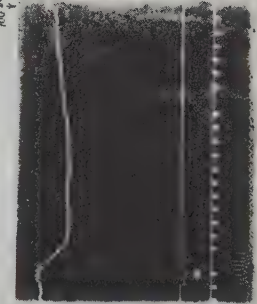


Fig. 19f

